A SYSTEMATIC APPROACH TO ABNORMAL CHEST IMAGES: RADIOGRAPHS AND COMPUTED TOMOGRAMS

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I. DEFINITIONS

- A. Pattern Seen on images
- B. Disease Seen on specimens
- C. Diffuse pulmonary disease

Nonspecific term for either destructive restrictive pulmonary disease involving all or most of the lung fields producing any pattern of opacity on chest films and CT.

D. Infiltrative lung disease

Nonspecific term for any restrictive pulmonary disease which infiltrates rather than destroys lung parenchyma.

E. Interstitial lung disease

Pathology term for thickening or destruction of pulmonary interstitium, which includes alveolar walls, septa and connective tissue surrounding bronchi and vessels (peribronchial and perivascular spaces). Correlates with functional term "restrictive lung disease" and with interstitial pattern on chest films.

F. Consolidative Pattern

Preferred over 'alveolar pattern' to describe the radiological pattern of filled air spaces.

II. PATTERNS

- A. Mass
- B. Consolidative
- C. Interstitial
 - 1. linear
 - 2. nodular
- D. Vascular
- E. Airway
 - 1. obstructive
 - 2. wall thickened

III. MASS

- A. Mechanism Local destruction of lung parenchyma
- B. Radiological Sign Any localized opacity not completely bordered by fissures or pleura
- C. Differential Diagnosis
 - 1. Malignancy Primary, metastatic, lymphomatous
 - 2. Granulomatous disease infectious or noninfectious, active or inactive.
 - 3. Other inflammation, including pneumonia and abscess
 - 4. Benign neoplasm
 - 5. Congenital abnormality
- D. Crucial appearance characteristics for inactivity
 - 1. Calcification Central, lamellar
 - 2. Evolution 2 year stability or regression
- E. Clinical variables
 - 1. Age
 - 2. Symptoms and signs
 - 3. Risk factors
 - Smoking
 - Occupation, exposure
 - Previous carcinoma
 - Concurrent diseases

IV. CONSOLIDATIVE (ALVEOLAR) PATTERN

A. Mechanism

produced in pure form only by alveolar filling, but may mimicked by alveolar collapse. airway obstruction, or rarely confluent interstitial thickening, or a combination of these.

B. Radiological signs

Fluffy, cloud-like, coalescent opacities which may have sharp edges when limited by fissures or pleura. Complete air bronchograms unless airways obstructed.

- C. Differential Diagnosis
 - 1. Hemorrhage Blood Embolism, trauma
 - 2. Exudate Pus Pneumonia, pneumonitis
 - 3. Transudate Water Congestion, ARDS
 - 4. Secretions Protein Mucous plugging, alveolar proteinosis
 - 5. Malignancy Cells Alveolar cell carcinoma, lymphoma

D. HRCT appearance – may be same density as blood or may be ground-glass GROUND-GLASS density favors ACTIVE process over chronic and MILD over severe

Mechanisms:

Consolidative pattern with diffuse aerated alveoli
Severe interstital pattern
Fatty material in consolidated alveoli
Obstructive pneumonitis or alveolar proteinosis
Vascular plethora – often mosaic

V. INTERSTITIAL PATTERN

A. Mechanism

Diffuse or irregular thickening of lung interstices or architectural destruction of interstitium (honeycomb or "end stage" lung).

- B. Radiological Signs
 - 1. Linear form

Characterized by reticulations (lines in all directions and by septal lines (Kerley lines).

2. Nodular form

Characterized by small, sharp, numerous, evenly distributed, uniform (especially uniform in shape) nodules

3. Destructive form

sole distinctive feature is peripheral, irregular formation

- C. Additional linear signs with CT (especially HRCT)
 - 1. Peribronchial thickening
 - 2. Perivascular thickening
 - 3. Alveolar wall thickening
 - 4. Subpleural thickening
 - 5. Thick-walled cystic spaces (honeycomb)
- D. Differential diagnoses:
 - 1. Linear form LIFE lines
 - Lymphangitic spread of malignancy
 - Inflammation
 - Fibrosis
 - Edema
 - 2. Nodular form
 - Granulomas
 - Hematogenous spread of malignancy
 - Pneumonoconiosis

3. Destructive form

- Fibrosing aveolitis (IPF, UIP)
- Specific causes of fibrosis

E. HRCT distribution of linear interstitial patterns:

- L central
- I diffuse or lower lungs
- F peripheral
- E central, with dilated vessels

F. Destructive form

Characterized by permanent lung destruction with architectural distortion of the parenchyma. Early appearance is nonspecific, usually identical with linear form. Late findings include peripheral cystic spaces ("Honeycomb" or "End Stage Lung") and irregular reticulations with volume loss and deformity of lungs. Most easily seen with HRCT.

VI. VASCULAR PATTERNS

A. Mechanism

Caused by increased, or decreased perfusion, altering diameter of pulmonary vessels.

B. Radiological Signs

Changes in diameter of specific vessels.

C. Common Examples:

- 1. Congestion engorged veins, especially upper lungs
- 2. Emphysema diminished vessels
- 3. Arterial hypertension large central arteries with peripheral tapering
- Thromboembolism locally diminished vessels with possible vessel mass centrally located
- 5. Shunt vascularity All vessels enlarged
- 6. Bronchial circulation Irregular vessels in unusual directions
- 7. Lymphangitic carcinoma Irregular infiltration around vessels may resemble vessel enlargement

VII. AIRWAY (BRONCHIAL) PATTERNS

A. Mechanism

Caused by complete or partial obstruction of airways or by thickening of airway walls.

B. Forms

- 1. Complete airway obstruction Produces opacity and decreased volume of lung distal to the site of obstruction. May resemble localized consolidation, but without air bronchograms
- 2. Partial obstruction Produces lucency and increased volume by air trapping
- 3. Wall thickening Tram tracks, central cystic spaces or circles

C. Differential Diagnosis

- 1. Opacities Endobronchial neoplasms, granulomas, inflammatory, benign or congenital masses, mucous plugs, foreign bodies.
- 2. Lucencies COPD, cysts, blebs, pneumatoceles
- 3. Thickening Bronchiectasis, chronic bronchitis

D. Additional Signs with CT

- 1. Thick-walled airways, circular on end, often "signet rings"
- 2. Cystic spaces centrally located
- 3. Cystic spaced with very thin walls or no apparent walls
- 4. Thin, stretched vessels
- 5. Bronchiectasis

E. Destructive Airway Disease - Bronchiectasis

- 1. Characterized by central (perihilar) cystic spaces, increased lung volumes and thickened airway walls (circles, when seen on end, and tram tracks)
- 2. Distinct from destructive interstitial disease (see V. E.), although some patients also have secondary interstitial changes, especially localized fibrosis.

F. Small Airway Disease

- 1. Often appears as "mixed pattern" of patchy opacities and lucencies, also called "alveolar nodules"
- 2. Usually includes bronchiolitis, producing complete and partial obstructions of small airways
- 3. Causes of bronchiolitis
 - a. Infectious eg. viral, mycoplasmal
 - b. Allergic
 - c. Toxic eg. chlorine, phosgene
 - d. Idiopathic
- 4. May thicken bronchial walls and resemble interstitial reticulations.

TABLE I

BASIC DIFFUSE PULMONARY PATTERNS

Consolidative
Interstitial
Linear
Nodular
Vascular
Airway

Obstructive Wall thickened

TABLE II

INTERSTITIAL PATTERNS

Differential Diagnosis

Linear form — LIFE lines
Lymphangitic spread of malignancy
Inflammation
Fibrosis
Edema

Nodular form

Granulomas

Hematogenous spread of malignancy

Pneumoconiosis

Destructive form — Honeycomb ("end stage") lung

TABLE III

CAUSES OF LINEAR INTERSTITIAL PATTERNS

Lymphangitic Spread

Metastatic malignancy

Primary malignancy (usually adenocarcinoma)

Lymphoma (rarely)

Sjögrens Syndrome (rarely)

Lymphangioleiomyomatosis (rare)

Inflammation

Infections

Viral

Mycoplasmal

Bacterial (usually early or mild)

Pneumocystis carinii

Collagen Vascular Disease

Rheumatoid arthritis

Polymyositis and dermatomyositis

Scleroderma (progressive systemic sclerosis)

Allergic alveolitis (hypersensitivity pneumonitis)

Drug reactions

Waldenström macroglobulinemia

Amyloidosis

Idiopathic ("usual interstitial pneumonitis")

Fibrosis

Following inflammation (see above)

Asbestosis

Radiation pneumonitis

? Talcosis

Neurofibromatosis

Edema

Cardiac

Renal

Fluid overload

Anemia

Table IV

CAUSES OF NODULAR INTERSTITIAL PATTERNS

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Granulomatous Diseases
      Infectious
             Tuberculosis
             Atypical mycobacterial diseases
             Fungal diseases, especially:
                   Histoplasmosis
                   Coccidioidomycosis
                   Blastomycosis (N. A. and S. A.)
                   Cryptococcosis
                   Sporotrichosis
             Bacterial diseases, especially:
                   Nocardiosis
                   Actinomycosis
      Non-infectious
             Sarcoidosis
             Vasculitis-granulomatosis diseases
                   Wegener's
                   Lymphocytic
                   Bronchocentric
                   Allergic (Churg-Strauss)
            Histiocytosis (eosinophilic granuloma) (LCG)
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Hematogenous Metastases

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Pneumoconioses
Granulomatous
Silicosis
Berylliosis
"Benign"
Coal Worker's Pneumoconiosis
Siderosis
Stannosis
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